casts in the urine. The blood pressure often falls except in those severe, terminal stages of chronic interstitial nephritis with all the typical complications.

We adopted this simple plan about eighteen months ago. A brief summary of the history of one of the first cases treated will give an idea of the results obtained.

CASE REPORT

Mrs. S. First seen December 12, 1920; age 64; weight 172 pounds. She complained of shortness of breath, dizzy spells, marked gastric and abdominal distress and a general run-down feeling. She had been taking salts each morning on the advice of her physician. The blood pressure was 210/110. Traces of albumin and hyaline casts were found in the urine. The patient was given a basic diet with the fats and sugars limited, because of her overweight. The H ion concentration of the urine was tested from time to time and found to be close to a pH of 7.0. She gradually lost weight, with no discomfort. The abdominal distress disappeared. The bowels moved normally. Some of the specimens of urine contained no albumin or casts, and the blood pressure fell as follows:

Dec. 12, 1920—blood pressure 210/110	weight 172	lbs.
Dec. 29, 1920—blood pressure 200/110	weight 171	lbs.
Jan. 12, 1921—blood pressure 195/110	weight 168	lbs.
Jun. 1, 1921—blood pressure 170/110	weight 148.25	lbs.
Nov. 1, 1921—blood pressure 172/115	weight 147.	lbs.

Patient stated that her bowels were moving normally, the gastric distress had disappeared, and that she was feeling better than she had felt for a long time.

Feb. 23, 1921—blood pressure 170/95	weight 160.5	lbs.
Apr. 20, 1921—blood pressure 160/110	weight 148.75	lbs.
Jun. 1, 1921—blood pressure 170/110	weight 148.25	lbs.
Nov. 1, 1921—blood pressurs 172/115	weight 147.	lbs.

Patient had returned from a summer in New York and Cape Cod, and had felt exceptionally well during the whole time she had been away.

April 26, 1922—blood pressure 160/105 weight 148 lbs.

Nurses Practicing Medicine in New England—Considerable interest has been aroused concerning the problem presented by nurses who have been treating injuries without having secured attendance by physicians. This matter has been frequently referred to by physicians who have felt that nurses have assumed responsibilities not warranted under our laws, and in some instances doctors have felt that the activities of nurses have in a very definite way invaded the field of medical practice, although the nurses have been sufficiently warned. Since the alleged practice of medicine by nurses has seemed to be on the increase there has been no reason why the authorities should decline to act. The Board of Registration in Medicine has tried to bring about compliance with the law without resorting to drastic measures, but reports of questionable methods have become so common and demands for action have been so insistent that it became necessary to report the facts in two recent cases to the prosecuting authorities. . . .

The whole question is complicated and delicate, but it can be solved by the nurses themselves, even though managers of industrial plants and some physicians try to induce nurses to practice medicine either as a convenience or to save expense.

The general purpose of the laws governing medical practice is to protect the patient from incompetent service. and nurses should observe the requirements because it is a law and also because sooner or later some nurse may attempt to render a service which is beyond her ability.—(Boston Medical and Surgical Journal, April 6, 1922.)

CARDIAC DYSPNOEA OR RATHER CARDIAC SHORTNESS OF BREATH

A QUESTION OF RESERVES

By HARRY SPIRO, M. D., San Francisco

In this discussion an attempt will be made to answer the following questions:

1. Is dyspnoea caused by diseased muscles or valves of the heart, and if so, why does not every patient with organic heart disease complain of it?
2. Is dyspnoea due to an edema of the alveolar tissue of the lung, and if so, is the edema always present? If it is, why does dyspnoea only show when the patient exercises and disappears almost immediately upon rest?
3. Is dyspnoea due to a lack of oxygen in the blood, and if so, why cannot these patients assimilate the oxygen of the air as well as any other person?
4. Is dyspnoea due to an improper exchange of CO_2 between the blood and the alveoli of the lungs, and if so, is this a heart or a lung fault?
5. Is dyspnoea due to an acidosis of the tissues or of the blood, and again what is the relation between the heart and acidosis?
6. Is dyspnoea due to a lack of tone or weakening, or partial paresis of the muscles of respiration like the diaphragm, and intercostals, and if so, why call it cardiac dyspnoea?

Dyspnoea, difficult breathing, or shortness of breath, will bring a patient to the physician more quickly than almost any other symptom, because it is a symptom which interferes so materially with his comfort, with his sense of well-being, and earning power. Difficult breathing is unaccountable to the patient. He may awaken in the morning feeling fine and refreshed, but the least effort makes him sit down, not because he is tired, but because he is short of breath. He may come home in the evening, eat a fairly hearty meal, and afterwards have an extraordinary amount of discomfort and shortness of breath, particularly so if he should attempt any exercise. Such, in brief, is the commencing of a chain of symptoms, which, as Facio says, are gradual, progressive and fatal. Just as this symptom is so important to the patient, so it at once becomes to the physician, because he knows that such symptoms are not imaginative. The physician recognizes dyspnoea as a distress signal, a clear call for help from the vital centers of our bodies. While it is true that shortness of breath may have underlying causes other than the heart, it is advisable to prove the heart innocent, particularly if the shortness of breath is in direct relation to moderate effort.

How is dyspnoea brought about? An individual with disease of the valves or muscles of the heart may go through life without knowing there is anything wrong. He may get sick, and get well again. He may eat, drink, smoke and work without much distress. Occasionally he does things to excess, and on such occasions the excess adds an "accumulating resistance" to the heart's action—accumulating because the additional strain has caused some partially degenerated or weakened heart muscle to give way completely, and on each such occasion a call is made on the reserve power

of the heart's muscle because it is the reserve power that has carried the defective heart to this point. If the heart hypertrophies properly—by each ventricle keeping its balance of power—well and good. If the reserve power becomes partially depleted, the strain may be greater on one or the other ventricle with the result that the hypertrophy is irregular, because one of the ventricles becomes out of proportion in power to the other.

One of the interesting episodes in the annals of medicine was the controversy between Welch and Sahli over the cause of pulmonary edema. As a result of extensive experiments both teachers were proved partially correct. Sahli claimed that edema of the lungs was caused only by some local irritant or poisoning or infection, and that if Welch was correct he was wrong anyway, because, as Sahli said, "Welch's theory did not apply to clinical cases." Welch had claimed that the edema could be caused by a disproportion between the powers of the two ventricles, resulting in a damming back of the blood in the lungs or improper circulation of blood in the lungs, and so edema was produced, and today we know that is a fact. The right ventricle may pump the blood fast enough into the lungs, but the left may not drain it out properly. If the left ventricle shows signs of distress the condition is recognized by simply feeling the radial pulse, but if the right side partially gives way, the symptoms are not so easily judged. Merely a cough and moderate cyanosis may be the only symptoms, or even if the patient is feeling fairly well, a few rales of different qualities at the base of the lungs may suggest a moderate edema of the lungs, but perhaps no severe dyspnoea yet, so we will go further, remembering how a weak heart produces edema of the lungs.

We will now consider lack of oxygen in the blood. One of the facts definitely proved during the war was that a very moderate edema of the lungs interferes materially with the proper absorption of oxygen. As with the reserve power of the heart muscle, so it is with oxygen. The blood retains a reserve supply of oxygen of about three times as much as is needed. Experiments by Lundsgaard, Steadie, and others show that the total oxygen carrying power of arterial blood is about twenty volumes per cent. We use about five and one-half per cent at rest, and perhaps ten to twelve per cent with severe effort. After rest the reserve supply is immediately replenished. If an obstruction occurs in the way of the oxygen's ready absorption, as is the case in edema of the lungs, the reserve oxygen gradually becomes depleted. The patient may go on for some time without a serious disturbance, as there is no actual lack of oxygen even after manifest injury to the lungs, because the tissues will absorb their quota of oxygen even though the supply is limited. Sooner or later, as the reserve nears exhaustion, the respiratory center in the brain takes note of it and sends out a call to the respiratory muscles for more work and faster work. Here again we see one of nature's resources, another of the reserve systems, this time called the pulmonary reserve. Actually this is the greatest amount of air the lungs can inhale after the greatest expiratory effort. Ordinarily, we utilize about five liters of air at ordinary respiration at rest per minute. This amount can be increased ten to twelve times without the person being unduly conscious of it. An increase to sixty or seventy liters is common, but there is always a return to the former level. However, in a patient with the above symptoms of insipient incompetence, resulting in a moderate edema of the lungs, the so-called threshhold is raised from five to twenty liters, and so is that much nearer the upper limits of the pulmonary reserve, and the reserve instead of being fifty or sixty liters per minute is now thirty or forty liters per minute, and so an effort that would pass unnoticed in a normal person would to one in this condition become noticed because of the increased respiratory effort needed.

The question of the actual method, not the underlying cause, of this depreciation of the pulmonary reserve or vital capacity is still under active investigation. Rooney of the Stanford cardiac clinic is now doing some work in this line, so I prefer not to discuss it, except to say that probably the air spaces may be encroached upon by the enlarged heart itself acting by compression of lung tissues and by the increased amount of blood in the lungs. The dilated veins may interfere with proper expansion of some air vesicles and so cause overexpansion of others. This, if true, may interfere with the proper draining of the residual air, and even though the respiratory rate is increased and the total air inhaled and exhaled is greater, still the oxygen is not properly absorbed. Now the respiratory center is calling on the respiratory muscles for heavier work, as a proper level of oxygen must be maintained. The respiratory muscles, while tired and probably weakened, still respond. Exhalation ceases to be a nearly passive effort and now is a severe effort, but a time arrives when the respiratory muscles produce more of the products of fatigue than the weakened heart muscles and lack of oxygen can eliminate or neutralize. This product of fatigue also is a stimulant to the already overexcited respiratory center, and the respiration on the least effort over the ordinary becomes quicker and more shallow—another link in nature's chain of defense has become weakened.

We are now in front of one of the last lines of defense which in time of development may be one of the first. It is placed last in the discussion because it is one of the last to break permanently. So far, all processes discussed were shown to depend on the question of reserves, each one of the different vital functions were shown to have tremendous reserves or the power to produce reserves, and as the reserves were encroached upon the patient's body was called upon for extra effort. In other words, extra effort takes the place of former reserves; the reserve we now face is the popular alkali reserve, the question of acidosis, the question of carbon dioxide interchange. This is called the internal respiration. Manifestly, it is impossible to condense the volumes that have been written on this subject into one or two pages.

Physiological and biological chemistry is deep water reasonably safe for Priestly, Heldane or Fisher, but not for most physicians. However, as we said, the internal respiration is the interchange between the tissue cells, the blood cells, and the blood plasma of CO_2 and oxygen, and certain acids and bases. CO_2 is an end product and is capable of forming H_2, CO_3. Now, the greater the accumulation of CO_2 in the blood, naturally the more acid the blood would become, if it were not for the great alkali reserve. The blood must not become acid; if it did, death would very quickly result. Nature has provided an almost permanent alkali level, and as a safeguard to this level has provided tremendous reserve alkali and also the power to produce reserve alkali. The symbol of the alkali reserve of the body can be taken as the sodium bicarbonates. If the CO_2 accumulates too fast so that the CO_2 pressure in the blood rises, a reaction takes place between the blood corpuscles and the sodium chloride in the plasma, resulting in an absorption of the chlorine to form H.C.L., which enters the red blood corpuscles, leaving sodium free. The sodium combines with the CO_2 and forms acid sodium carbonate, so that instead of an acid condition, we really have an alkali condition in the blood; but before this interchange has taken place the CO_2 has stimulated the hypersensitive respiratory center, which calls upon the respiratory muscle for renewed efforts, and large amounts of the CO_2 are immediately eliminated. The kidneys also eliminate large amounts of the bicarbonate that is formed, so that the alkali level which was temporarily raised is again equalized, but the reserve is lessened. The chlorine may now leave the red blood corpuscles and again combine with the soda forming Na Cl., and thus setting free enough CO_2 to maintain its proper level. The CO_2 level is as important as the oxygen level, and if the CO_2 is decreased too greatly it may not return to its proper level, thereby causing disaster. Reserve alkalies having been washed out by the kidneys—an ordinary effort that might pass unnoticed—calls for extra stimulation by the respiratory center, and as there is not enough alkali reserve on hand it must manufacture alkali for the occasion, and so we have the dyspnoea of effort—easily produced because of lack of reserve.

The chemists have experimentally proved that a proper oxygen level must be maintained or death result, and death results even though the oxygen is brought up again to its former level. A CO_2 level also must be maintained. Upward tendencies of the CO_2 level are equalized by the respiratory apparatus. If the level descends below a safe point, it cannot be again raised even by giving CO_2, and a Haemolysis takes place and death results.

There also is an alkali level. If this falls too low, nature reproduces alkali. We should not feed alkalies when nature does not want it, even though there is a lack of alkalies. It is the level that is of utmost importance. If the alkali level is without reserve, or reserve power partially depleted, it would be of use to furnish alkalies, provided we could regulate the respiratory apparatus so as not to reproduce the results that brought on the original trouble. The loss of alkali is not the cause—it is the lack of oxygen that started the trouble.

With the above thoughts before us we return for a moment to the question of diagnosis and treatment. Every case of dyspnoea is not due to the heart, and every case of heart disease need not have constant dyspnoea. Effort can produce dyspnoea in a person whose heart is sound, but dyspnoea is one of the first and most reliable symptoms that the heart is diseased, and in a case of mild dyspnoea a diagnosis can, as a rule, be made because the physical examination can be made leisurely and the defect in the heart proved. But how about the sudden alarm at night, and you walk in on a patient sitting or propped up in bed, as a rule pale or slightly cyanotic, and covered with perspiration, breathing rapidly and labored, foam coming from the mouth, a pulse fast and sometimes irregular? This may be a first attack, and if that is the history obtained from the family, it most likely is a heart attack and should be treated as such, particularly if the attack is attributed to some excess of food or work. Compare this to ordinary asthma bronchial in type. This is generally seasonal, and while there must be a first attack, it is usually long before a physician is called. The patient usually does a great deal of the talking. He is very sick, but does not impress the physician that he is desperately ill. Between his gasps and his wheezes he tells of repeated attacks of bronchitis on exposure to weather as the cause, and after being relieved will still cough up large amounts of purulent sputum. The expiratory effort in bronchial asthma is prominent. Both inspiration and expiration are difficult in cardiac asthma and the rate faster. If the attack stops suddenly, as it may, the patient may sleep and awaken apparently cured, or he may have a severe bronchitis as a result of the exposure during the attack. This is dangerous because it is easy to neglect the heart and think that after all the patient did have a real bronchial asthma. The next attack, if treated as ordinary asthma, may result in sudden death.

The treatment of cardiac dyspnoea is first rest, and more rest. Oxygen used vigorously is valuable. Haldane and Priestly have proved that recoveries from lung edema by the use of oxygen occur where proper methods of administration are employed. Digitol, digitaline, tincture of digitalis are indicated and should be given to the physiological limit. It is advisable to be careful of adrenalin. Atropin blocks the vagus, and thus increases the difficulties in lowering the heart rate. Strychnine, camphorated oil, whisky and caffine are helpful and do not interfere with digitalis. Morphine and oxygen given together are helpful. Without oxygen, morphine is moderately dangerous, because it depresses the respiratory center which must be kept up to its work.

Some patients may die quietly and peacefully

after administration of alkalies while others die in convulsions, with or without alkalies. This method of treatment, therefore, is of questionable value. McLeod advises giving alkalies and testing the urine to determine if acidosis is present. Whitney warns against this procedure and with good judgment. He advises giving a diuretic or a physic at the same time. Too much alkali should not be given, particularly if the kidneys are defective, because the same conditions caused by surplus of acid can also be caused by too much alkali. Keeping the urine neutral is probably the best procedure. The patient should be kept in a semi-reclining position because reclining may embarrass the right heart. The lungs should be stretched by breathing exercises for a few moments several times a day. Caution is necessary to avoid overexercising, as it is possible to embarrass the right heart and right coronary artery. Epsom salts is one of the best laxatives if the kidneys are functioning properly. For a very large and full-blooded patient, venesection is sometimes a good procedure. Even though it reduces the oxygen carrying power of the blood it may relieve an overstretched vascular system.

A simple, though not absolutely reliable, test for acidosis is to give bicarbonate of soda until the urine becomes alkaline, and then watch the amount of bicarbonate necessary to keep the urine neutral. Less than four grams per day is normal and safe to use if neutral red or sulphophenolthalein is used as an indicator.

212 Stockton Street.

THE SLANDERER

The very name invokes loathing. Though more or less in human form, this degenerate remnant of the silurian age is the most contemptible of creatures. The scandalmonger is disliked, the liar is despised, but the slanderer is loathed. Using falsehoods or facts that are distorted as some would juggle statistics, the slanderer spreads a most subtle poison that blasts lives and reputations. Slander cannot be controlled any more than you can stop a lie, once it has gained credence. Compared with the social diseases, it is the greatest evil of our age. The slanderer is more dangerous and despicable than those misguided enemies of society who use bombs and poison secretly.

Whenever you discover a slanderer posing as an honorable member of our profession, let your conscience be your guide, but be sure you do your full duty.—Malsbary, editorial in the Southern California Practitioner.

Speaking of Babies—According to George Ade (Cosmopolitan, February) the old saying that "the hand that rocks the cradle rules the world" is now changed to "the hand that rocks the cradle should be amputated." There is more of humor and interesting philosophy in the article.

"The human race is, according to a guess by H. G. Wells, 75,000 years old, and only in the last twenty-five years has any one known how to welcome a baby and take care of it. All those who survived during the preceding 74,975 years were just plain lucky.

"The first impression gained by a baby of the nineteenth century must have been that Earth is inhabited by elderly people, all females except one and all wearing spectacles.

"The newly arrived of 1921 probably decides that the world is walled with white, tinctured with anesthetics and peopled by efficient internes and calm nurses."

THE PLACE OF PATHOLOGY IN THE PRACTICE OF MEDICINE *

By WALTER V. BREM, M. D., Los Angeles

If one should confine himself strictly to the subject of this paper, his task would be indeed a gratuitous one, for there is certainly no one in touch with the progress of medicine who does not recognize the practical assistance that the pathological laboratory may give to a clinician. There are, of course, some clinicians who have become discouraged in the use of laboratories by reason of incompetent service and because of the fact that ill-informed or overenthusiastic laboratory workers have advanced too often unwarranted claims that could not be fulfilled.

On the other hand, there are many clinicians who are oversanguine regarding the service that a laboratory may render, and others to whom laboratory work is like a simple test tube experiment, that he who runs may read. The former do not appreciate the limitations of the laboratory, and the latter do not understand the difficulties of the work and the necessity of its being directed by an experienced pathologist.

SCOPE OF PATHOLOGY

Pathology, in its broad sense, is the study of the causes of disease, of the structural changes (macroscopic, microscopic and chemical) underlying disturbed function, and of objective manifestations of altered structure and perverted function. Pathology is not limited, as is so often assumed, to autopsies and the microscopic examination of tissues, but the term covers the underlying conditions and objective manifestations of disease. A fairly good foundation in pathology is, therefore, a fundamental prerequisite for a clear conception of disease, and it is as essential for a clinician to be able to visualize common structural changes and to think in terms of pathology, as it is for the pathologist to have had sufficient clinical experience to hold in his view the welfare of patients.

SELECTION OF A PATHOLOGIST

Having acquired a good fundamental knowledge of pathology, so that he knows what examinations are indicated in given conditions, the busy clinician is confronted with the problem of how to secure the pathological service needed in his practice. No longer can his own office encompass the vast field, no longer can he himself find time to master the technique and perform the complicated tests indicated, and no longer can he train his own technician and supervise the work except in a very limited way. Out of this need the modern clinical laboratory has developed, with its departments of tissue pathology, bacteriology, serology, biochemistry and clinical microscopy. The laboratory is no longer limited to tests of urine for albumin, examinations of sputum for tubercle bacilli, and blood counts (relatively simple tests in the department of clinical microscopy), but its work deals with the essential

* Address in the General Session of the Annual Meeting of the Medical Society of the State of California, Yosemite Valley, May 15-18, 1922.